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Planting Density Influences Disease Incidence and Severity of Sclerotinia Blight in Peanut

Andrea L. Maas,* Kenton E. Dashiell, and Hassan A. Melouk

ABSTRACT

Sclerotinia blight, caused by *Sclerotinia minor* Jagger, has become one of the major limiting factors in peanut (*Arachis hypogaea* L.) production. The objectives of this research were to evaluate the effects of plant spacing on disease incidence and severity of Sclerotinia blight in peanut research plots, to measure the level of apparent resistance at different seeding rates, and to determine which methods would produce clearest selection criteria in space-planted breeding plots. Four peanut cultivars, Tamspan 90, Southwest Runner, Okrun, and Flavor Runner 458, were evaluated in field plots at four plant spacings (6, 15, 30, and 46 cm) in 2003 and 2004. Increased plant spacing improved sensitivity of disease incidence based determination of cultivar resistance but did not increase mean incidence significantly. Disease severity reached the highest level at the widest plant spacing. Final disease incidence provided excellent differentiation of genotypes with different levels of resistance and required the least amount of labor as compared with other methods of disease assessment.

THERE ARE many constraints to peanut production, including a wide array of insects, diseases, and abiotic stresses. Sclerotinia blight has become one of the major limiting factors in peanut production (Melouk and Shokes, 1995). The first report of Sclerotinia blight affecting peanuts in the USA was in Virginia in 1971. In recent years, the disease has become more severe and spread to North Carolina, Oklahoma, New Mexico, Louisiana, and Texas (Smith et al., 1991a; Wildman et al., 1992). Yield losses of 10% are not uncommon; however in cases of severe infection, yield losses of up to 50% may occur in a single field (Melouk and Shokes, 1995).

Sclerotinia minor will attack all tissues of the peanut plant, but stem infections are the most economically important because reproductive pegs are attached to the stems (Chappell et al., 1995). Temperature, relative humidity, and soil moisture play vital roles in the infection and colonization of plant tissues by *S. minor. Sclerotinia minor* is a soil-borne pathogen. The disease is most severe during cool, wet weather, with an optimum growth range of 15 to 25°C and a relative humidity approaching saturation (95–100%). High humidity promotes myceliogenic germination of sclerotia and is positively correlated with disease development (Dow et al., 1988a, 1988b). Disease development in the field is low when plants are small and without a dense canopy or complete ground cover. Outbreak of Sclerotinia blight is most often observed after vines are within 15 cm of touching or after vines lap between rows (Dow et al., 1988b; Phipps, 1995). Sclerotinia blight development is greatest as the plants reach maturity in September and October because of cooler night time temperatures and higher relative humidities normally associated with fall climate changes. During this time the plant canopies expand, contributing to the maintenance of higher humidity close to the ground (Dow et al., 1988b).

Current Sclerotinia blight management recommendations include planting resistant cultivars, avoiding high seeding rates, cultivating before 15 June or eliminating cultivation entirely by integrated pest management to reduce the negative effects of nontarget fungicide applications, weekly field scouting for early detection, and fungicide treatments (Brenneman et al., 1988). "Omega 500F" (SCP 71512–1B-1000 0503 126357, Syngenta, Greensboro, NC), a new generation fluazinam (Smith et al., 1991a), has been effective for control of Sclerotinia blight in peanut; however, treatments are costly, particularly with the reduced prices associated with the elimination of the peanut quota system (K.E. Dashiell, personal communications, 2004). *Sclerotina minor* has a wide range of hosts including 21 families, 66 genera, and 94 species of both cultivated and wild plants, surviving 3 to 8 yr in the soil as sclerotia without a host (Abawi et al., 1985; Goldman et al., 1995; Melzer et al., 1997). Wide host ranges and sclerotial longevity limit the effectiveness of crop rotation as a means of control for Sclerotinia blight (Goldman et al., 1995).

Use of host plant resistance is generally included as the primary means of mitigating production losses in grower fields, particularly when the commodity prices are low (Jordan et al., 1999). A single study published in 1992, utilized area under the disease progress curve (AUDPC) of disease severity to study resistance heritability. This study indicated that while broad sense heritability was high (41–50.3%) narrow sense heritability was low (14–23%) (Wildman et al., 1992). There seem to be multiple mechanisms of resistance that control infection by *S. minor*. These factors include avoidance of disease due to architecture, maturity, and/or greater resistance of the plant tissue (Chappell et al., 1995). Genotypes with more prostrate growth habits exhibit more susceptibility to disease than those with a more upright growth habit because of increased plant canopy moisture and reduced temperatures. Detached-shoot tests have demonstrated that there is also an additional physiological form of resistance of an unknown form (Akem et al., 1992). Peanut breeding lines with spanish ancestry appear to be more resistant to *S. minor* than other market classes because of the more upright architecture which

A.L. Maas, USDA-ARS, Coastal Plain Exp. Sta., P.O. Box 748, Tifton GA 31794; K.E. Dashiell, USDA-ARS, Northern Grain Insects Res. Lab., 2923 Medary Ave., Brookings, SD 57006; H.A. Melouk, USDA-ARS, 127 NRC, Oklahoma State University, Stillwater, OK 74078. This manuscript is a portion of the senior author's dissertation required for the Ph.D. degree at Oklahoma State University. All programs and services of the USDA and Oklahoma State University are offered on a nondiscriminatory basis without regard to race, color, national origin, religion, sex, age, marital status, or handicap. Received 7 Oct. 2005. *Corresponding author (amaas@tifton.usda.gov).

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677 S. Segoe Rd., Madison, WI 53711 USA

increases air and light penetration in the plant canopy (Wildman et al., 1992). The objectives of this research were to evaluate the effects of plant spacing on disease incidence and severity of Sclerotinia blight in peanut research plots, to measure the level of apparent resistance at different seeding rates, and to determine which methods would produce clearest selection criteria in space-planted breeding plots.

MATERIALS AND METHODS

Four peanut cultivars were evaluated for Sclerotinia blight incidence and severity in small field plots at four plant spacings, 6 cm (75 seeds/4.57 m), 15 cm (30 seeds/4.57 m), 30 cm (15 seeds/4.57 m), and 45 cm (10 seeds/4.57 m), in 2003 and 2004. Four cultivars, Tamspan 90, Southwest Runner, Okrun, and Flavor Runner 458, were used in this study, because they exhibit widest separation of resistant and susceptible types for Sclerotinia blight grown in Oklahoma.

Tamspan 90 is a spanish market type with moderate resistance to Sclerotinia blight (Smith et al., 1991b). Southwest Runner is a runner market-type peanut cultivar with moderate resistance to *S. minor* comparable to Tamspan 90 (Kirby et al., 1998). Okrun was included as a susceptible (Banks et al., 1989). Flavor Runner 458 is a Sclerotina blight susceptible high oleic runner type cultivar (Horn et al., 2001).

Plots were established at the Caddo Research Station near Fort Cobb, OK, and infested with 3.3 g m^{-1} of inoculum in 2003 when sclerotia density was below one sclerotia $(100\ g)^{-1}$ of soil. Plots were not artificially infested in 2004 because of early season disease onset. *Sclerotinia minor* was grown on sterilized oat seeds which were inoculated with 3- to 4-d-old cultures grown on potato dextrose agar. Oat cultures were grown for 2.5 to 3 wk until sclerotia formed. Cultures were then spread flat and allowed to bench dry for an additional 3 to 4 wk. The dried inoculum was spread over plots at 3.3 g m^{-1}. Mean low ambient temperature was 17°C for both 2003 and 2004; mean high temperatures were 30 and 29°C for 2003 and 2004, respectively, for the months of May through October. Total rainfall was 37 cm in 2003 and 43 cm in 2004 for the months of May through October. The soil was a moderately deep, well drained loamy soil, nearly level to slightly sloping of the Cobb soil series.

A randomized complete block experimental design with split plots and four replications was used each year. Main plots were seeding rates and subplots were cultivars. Each block consisted of 16 two-row plots, 4.6 m long with rows 0.9 m apart, and a 1.5-m alley between the ends of plots. Stands were planted at desired spacing, 6 cm (75 seeds/4.6 m) which was the control used in grower fields, 15 cm (30 seeds/4.6 m), 30 cm (15 seeds/4.6 m), and 46 cm (10 seeds/4.6m). Planting was 20 May 2003 and 11 May 2004. Plants were scored and harvested on 17 Oct. 2003 and 6 Oct. 2004, allowing an average of 148 growing days. Recommended standard production practices for fertilizer, herbicide, and irrigation for Oklahoma were followed for both years (Oklahoma State University, 2000). Leaf spot was controlled each year with Headline (BASF, Research Triangle Park, NC) and Folicur (Bayer CropScience, Research Triangle Park, NC) fungicides.

Plots were evaluated and recorded biweekly for disease onset. Sclerotinia blight was first noted on 19 Sept. 2003 and 9 Aug. 2004. Disease onset in a plot was the first day on which any disease symptoms were evident.

Disease incidence was determined as the percentage of plants with above-ground symptoms. A plant having any evidence of Sclerotinia blight was scored as infected. Plots were scored a day before harvest each season with those plants dead because of other diseases eliminated from the incidence and severity scorings. Disease severity was calculated in two ways: (i) as the number of symptomatic primary lateral stems and main stems per plot divided by number of infected plants per plot and (ii) as the number of symptomatic lateral stems and main stems per plot divided by number of plants per plot. Generalized least squares were used to calculate means of disease incidence and severity among and within genotypes. Additionally, correlations of disease $severity_1$ with disease $severity_2$ and disease $severity_2$ with disease incidence were made by Proc Corr (SAS Institute, 2003). Unless otherwise indicated, a significance level of $p \leq 0.05$ was used to determine significant differences between treatments. The model used to compute significant differences and interactions was:

$$Y = \mu + \alpha_I + \beta_{j(i)} + \gamma_k + \tau_l + (\alpha\gamma)_{ik} + (\alpha\tau)_{il} + (\gamma\tau)_{kl} + (\alpha\beta\gamma)_{ij(i)k} + (\alpha\beta\tau)_{ij(i)l} + (\alpha\gamma\tau)_{ikl} + e_{ij(i)kl}$$

Where μ is the overall mean, α_i is the random effect of year i, $\beta_{j(i)}$ is the random effect of blocks nested within year i, γ_k is the fixed effect of spacing k, and τ_l is the fixed effect of cultivar l. Interactions evaluated were $(\alpha\gamma)_{ik}$, the fixed interaction effect of year i and spacing k, $(\alpha\tau)_{il}$ the fixed interaction effect of year i and cultivar l, $(\gamma\tau)_{kl}$ the fixed interaction of spacing $_k$ and cultivar $_l$, $(\alpha\beta\gamma)_{ij(i)k}$ the random effect of block j and spacing k nested within year i, $(\alpha\beta\tau)_{ij(i)l}$ the random effect of block j and cultivar l nested within year i, $(\alpha\gamma\tau)_{ikl}$ the fixed effect of spacing k, and cultivar l nested within year I, and $e_{ij(i)kl}$ as the experimental error.

RESULTS AND DISCUSSION

Disease initiation between the two years was 41 d apart, with first wilting noted in 2003 on 19 September and in 2004 on 9 August. This occurred because of an unusually cool weather pattern that provided for an average high of 27°C and low of 19°C for the dates of 5 to 8 Aug. 2004. Typical average highs of 40°C and average low of 24°C was observed during the same period in 2003. A significant increase from 2003 to 2004 of $p \leq 0.05$ for disease incidence measures and $p \leq 0.01$ for both disease severity measures because of this anomalous weather pattern was observed. This increase also supports the findings of Phipps (1995) that found weekly scouting and application of fungicides at the first appearance of disease rather than a fixed date of first application was most appropriate.

Sclerotinia blight was present in plots. Disease incidence over both years in individual plots ranged from 6 to 99%. Disease incidence for the susceptible lines ranged from 50 to 99% for Flavor Runner 458 and 66 to 99% for Okrun. Disease incidence values for the two resistant lines were 21 to 49% for Southwest Runner and 6 to 36% for Tamspan 90. A spacing × cultivar interaction was not observed for disease incidence ($p \leq 0.75$), indicating that classification for disease resistance was not affected by plant spacing (Table 1). Significant interaction of year × spacing ($p \leq 0.01$), year × cultivar ($p \leq 0.01$), and year × replication × spacing ($p \leq 0.01$) were expected because of the quantitative nature of the disease resistance (Wildman et al., 1992) and the large degree environment influences the infection process of *S. minor* (Dow et al., 1988a; Dow et al., 1988b; Phipps,

Table 1. Analysis of variance Sclerotinia blight for percentage disease incidence of peanut in 2003 and 2004 trials near Fort Cobb, OK.

Source of variation	Degrees of freedom	Sum of squares	Mean square	*F*	*P*
Year (Y)	1	645.6	645.6	4.2	*
Replication (*R*)	6	4363.1	724.2	4.8	**
Spacing (S)	3	5660.5	1886.8	12.55	**
Cultivar (C)	3	131 526.2	43 842.1	291.6	**
Y × S	3	2985.7	995.2	6.62	**
Y × C	3	8440.4	2813.5	18.7	**
S × C	9	874.9	97.2	0.65	NS
Y × R × S	18	9484.4	526.9	3.5	**
Y × R × C	18	4621.2	256.7	1.71	NS
Y × S × C	9	1251.4	139	0.92	NS
residual	54	8118.2	150.3		

* Significant at the 0.05 probability level.
** Significant at the 0.01 probability level.
NS for not significant.

Table 3. Analysis of variance Sclerotinia blight as an average number of infected stems per infected plant per two row plot of peanut in 2003 and 2004 trials near Fort Cobb, OK.

Source of variation	Degrees of freedom	Sum of squares	Mean square	*F*	*P*
Year (Y)	1	65.8	65.8	17.3	**
Replication (*R*)	6	88.3	14.7	3.9	**
Spacing (S)	3	14.8	4.9	1.3	NS
Cultivar (C)	3	168.7	56.2	14.8	**
Y × S	3	33.3	11.1	2.9	*
Y × C	3	5	1.8	0.48	NS
S × C	9	63.8	7.1	1.8	NS
Y × R × S	18	69.1	3.8	1.0	NS
Y × R × C	18	77.6	4.3	1.1	NS
Y × S × C	9	59.6	6.6	1.7	NS
residual	54	205.8	3.8		

* Significant at the 0.05 probability level.
** Significant at the 0.01 probability level.
NS for not significant.

1995). Disease incidence did not significantly ($p \leq 0.05$) increase with increased plant spacing within cultivars. Mean separations among genotypes did increase from $p \leq 0.05$ to $p \leq 0.01$ with increased plant spacing (Table 2), indicating increased plant spacing may improve differentiation of resistant from susceptible cultivars. This therefore suggests that individual plant selections could be made in breeding nurseries which are commonly planted on wider plant spacings with minimal apparently resistant plants chosen. For the 2 yr of this study, the lowest mean disease incidence for susceptible cultivars planted at 31 and 46 cm was 87%, indicating that the chance of selecting an escape with 2 yr of testing would be about 2%. The solid separation of plant disease response utilizing disease incidence for assessment suggests that the labor intensive disease progress methods previously utilized (Coffelt and Porter, 1982; Goldman et al., 1995) may not be necessary for evaluation of plant resistance, which supports the findings of Akem et al. (1992) which made comparisons of disease incidence and area under the disease progress curve methods at single plant spacing.

A spacing × cultivar significant interaction ($p \leq 0.08$) was not observed for disease severity$_1$, when considering only diseased stems per diseased plant, indicating that classification for disease resistance was not affected by plant spacing (Table 3). As this interaction was nearly significant, an additional year of data, to mitigate the effects of the unusually early disease onset in 2004, might result in a significant interaction. Disease severity$_1$, produced significant differences ($p \leq 0.05$) only at a plant spacing of 46 cm sufficient to allow all resistant types to be differentiated from all susceptible types (Table 4). Resistant cultivars ranged from 1.4 stems per plant to 4.1 stems per plant while the range for susceptible cultivars was 1.8 stems per plant to 7.8 stems per plant for all observations. Only Flavor Runner 458 had a significant difference ($p \leq 0.05$) within cultivar for plant spacing which was between the control at 2.4 stems per plant and 46 cm at 6.3 stems per plant, all others were not significant (Table 4). The only year interaction that was significant was year × spacing ($p \leq 0.04$) which would indicate failures of measurement were attributable to variability of disease assessment method and not yearly variation in environment (Table 3). The differential inoculum densities associated with soil-borne pathogens, the quantitative nature of this resistance, and plant architectural response contributed sufficient variability to make cultivar separations difficult using individual plant disease severity as a measure of disease resistance.

A significant interaction ($p \leq 0.004$) for spacing × cultivar was observed for disease severity$_2$, when diseased stems per total number of plants in the plot were considered, indicating that classification for disease resistance was affected by plant spacing (Table 5), which is

Table 2. Overall means by cultivar and plant spacing of Sclerotinia blight for percentage disease incidence of peanut in 2003 and 2004 trials near Fort Cobb, OK.

	Plant Spacing (cm)			
Cultivar	6.1	15.3	30.3	45.7
	%			
Flavor Runner 458	73.2 aA	85.8 aA	86.6 aA	93.5 aA
Okrun	81.7 aA	86.6 aA	97.67 aA	96.9 aA
Southwest Runner	25.9 bA*	28.2 bA*	36.7 bA**	37.2 bA**
Tamspan 90	8.4 bA**	11.2 bA**	17.8 bA**	31.2 bA**

* Significant at the 0.05 probability level for rate within a cultivar given as an uppercase letters (rows) and among lines for a given seeding rate given by lowercase letter (columns).
** Significant at the 0.01 probability level for rate within a cultivar given as an uppercase letters (rows) and among lines for a given seeding rate given by lowercase letter (columns).

Table 4. Overall means by cultivar and plant spacing of Sclerotinia blight as an average number of infected stems per infected plant per two row plot of peanut in 2003 and 2004 trials near Fort Cobb, OK.

	Plant Spacing (cm)			
Cultivar	Control	15.3	30.3	45.7
Okrun	5.2 aA	5.0 aA	5.9 aA	5.9 aA
Flavor Runner 458	2.4 bA*	4.1 abAB*	4.9 abAB*	6.3 aB*
Southwest Runner	3.5 abA*	2.9 abA*	3.3 bA*	3.5 bA*
Tamspan 90	3.2 abA*	2.3 bA*	2.2 cA**	1.9 bA*

* Significant at the 0.05 probability level for rate within a cultivar given as an uppercase letters (rows) and among lines for a given seeding rate given by lowercase letter (columns).
** Significant at the 0.01 probability level for rate within a cultivar given as an uppercase letters (rows) and among lines for a given seeding rate given by lowercase letter (columns).

Table 5. Analysis of variance Sclerotinia blight as an average number of infected stems per total plants per two row plot of peanut in 2003 and 2004 trials near Fort Cobb, OK.

Source of variation	Degrees of freedom	Sum of squares	Mean square	*F*	*P*
Year (Y)	1	30.6	30.6	24.4	**
Replication (*R*)	6	27.9	437	3.7	**
Spacing (S)	3	53.3	17.8	14.2	**
Cultivar (C)	3	360.8	120.3	96.1	**
Y × S	3	5.9	2.0	1.6	NS
Y × C	3	42.9	14.3	11.4	**
S × C	9	35.7	4.0	3.2	**
Y × R × S	18	23.2	1.3	1.0	NS
Y × R × C	18	35.1	2.0	1.6	NS
Y × S × C	9	9.2	1.0	0.8	NS
residual	54	67.6	1.3		

* Significant at the 0.05 probability level.
** Significant at the 0.01 probability level.
NS for not significant.

consistent with the increased differences between cultivars as spacing increased for disease incidence and disease severity (Table 6). Disease severity$_2$ also produced a significant increase in severity within genotype for the two susceptible cultivars, at a significance level of $p \leq 0.01$ between the control of 6 cm and the two highest spacing of 30 and 46 cm and $p \leq 0.05$ for 6.1 versus 15 cm. There was no significant difference among cultivars at 6 cm; however, at 15 cm, significance of $p \leq 0.05$ was shown and $p \leq 0.01$ at 30 and 46 cm between the resistant and susceptible cultivars (Table 6). Resistant cultivars ranged from 0.1 to 2.6 stems per plant, whereas susceptible cultivars ranged from 0.7 to 9.8 stems per plant. Significant interaction of year × cultivar ($p \leq 0.0001$) was obtained (Table 5), which may have been due to the quantitative nature of the disease resistance (Wildman et al., 1992; Wildman et al., 1992) and the large degree environment influences the infection process of *S. minor* (Dow et al., 1988a; Dow et al., 1988b Phipps, 1995) also seen with disease incidence scoring. These results indicate a final disease severity scoring that includes all plants in a plot and will allow for significant separation of resistant from susceptible cultivars when planted at 15-, 30-, or 46-cm spacing.

A correlation of 0.73 with a significance of $p \leq 0.0001$ was obtained for disease severity$_1$ and disease severity$_2$.

Table 6. Overall means by cultivar and plant spacing of Sclerotinia blight as an average number of infected stems per total plants per two row plot of peanut in 2003 and 2004 trials near Fort Cobb, OK.

	Plant Spacing (cm)†			
Cultivar	6.1	15.3	30.3	45.7
	%			
Flavor Runner 458	2.7 aA	4.3 aB*	4.9 aB**	5.0 aB**
Okrun	1.8 aA	3.5 aB**	4.8 aC*	5.4 aC**
Southwest Runner	0.8 aA	0.8 bA*	1.1 bA**	1.4 bA*
Tamspan 90	0.3 aA	0.3 bA*	0.4 bA**	0.6 bA**

* Significant at the 0.05 probability level for rate within a cultivar given as an uppercase letters (rows) and among lines for a given seeding rate given by lowercase letter (columns).
** Significant at the 0.01 probability level for rate within a cultivar given as an uppercase letters (rows) and among lines for a given seeding rate given by lowercase letter (columns).

A correlation of 0.80 with a significance of $p \leq 0.0001$ was obtained for disease severity$_2$ and disease incidence. Disease severity$_1$ was basically severity of a single plant where as disease severity$_2$ included total plot disease incidence as a component of measurement, which would explain the higher correlation of severity$_2$ with disease incidence than severity$_1$. The high correlation obtained among the severities and with disease incidence demonstrates that all three methods are measuring the same basic plant response. It is therefore appropriate to select the method with the clearest separation of phenotype.

Resistance scoring was demonstrated to be most defined when disease incidence was used and least defined when single plant severity was used, which indicates measures of resistance should be based on incidence rather then severity. Resistance scoring also indicated that increasing plant spacings to 15, 30, or 45 cm provided a clear delineation between resistant and susceptible cultivars in a single final assessment without the encumbrance of the multiple disease assessments required by area under the disease progress curve. The soil-borne nature of Sclerotinia blight resulted in an uneven distribution of inoculum sufficient to make a single plant disease severity assessment at any spacing less then 45 cm nonsignificant. The results presented in this paper support the findings of Phipps (1987, vol. 2, p. 52) who found no significant effect of seeding rate on final disease incidence. Dow et al. (1988a) thinned after bloom to prevent compensation and found that while thinning reduced disease incidence and severity, yield was also reduced it. On the basis of the findings reported here, lower plant densities should allow for sufficient assessment of disease in research plots, but reduced seeding rates in grower fields would not be appropriate.

CONCLUSIONS

Date of initial disease onset supports the notion that weekly scouting and application of fungicide at the first appearance of disease is appropriate for the southern Great Plain region. Disease incidence and severity tended to increase with increased plant spacing in susceptible cultivars. Calculations of disease severity that included all plants present in the plot relative to total diseased stems produced the most significant differences ($p \leq 0.01$) at the widest plant spacing between susceptible and resistant cultivars. Differences in final disease incidence were also significant ($p \leq 0.05$) and required less labor to evaluate relative to other methods of disease intensity measurement.

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